



NEUR 1P90
Library Seminar
Fall 2023 term

Ian Gordon



Ian Gordon, Teaching & Learning Librarian

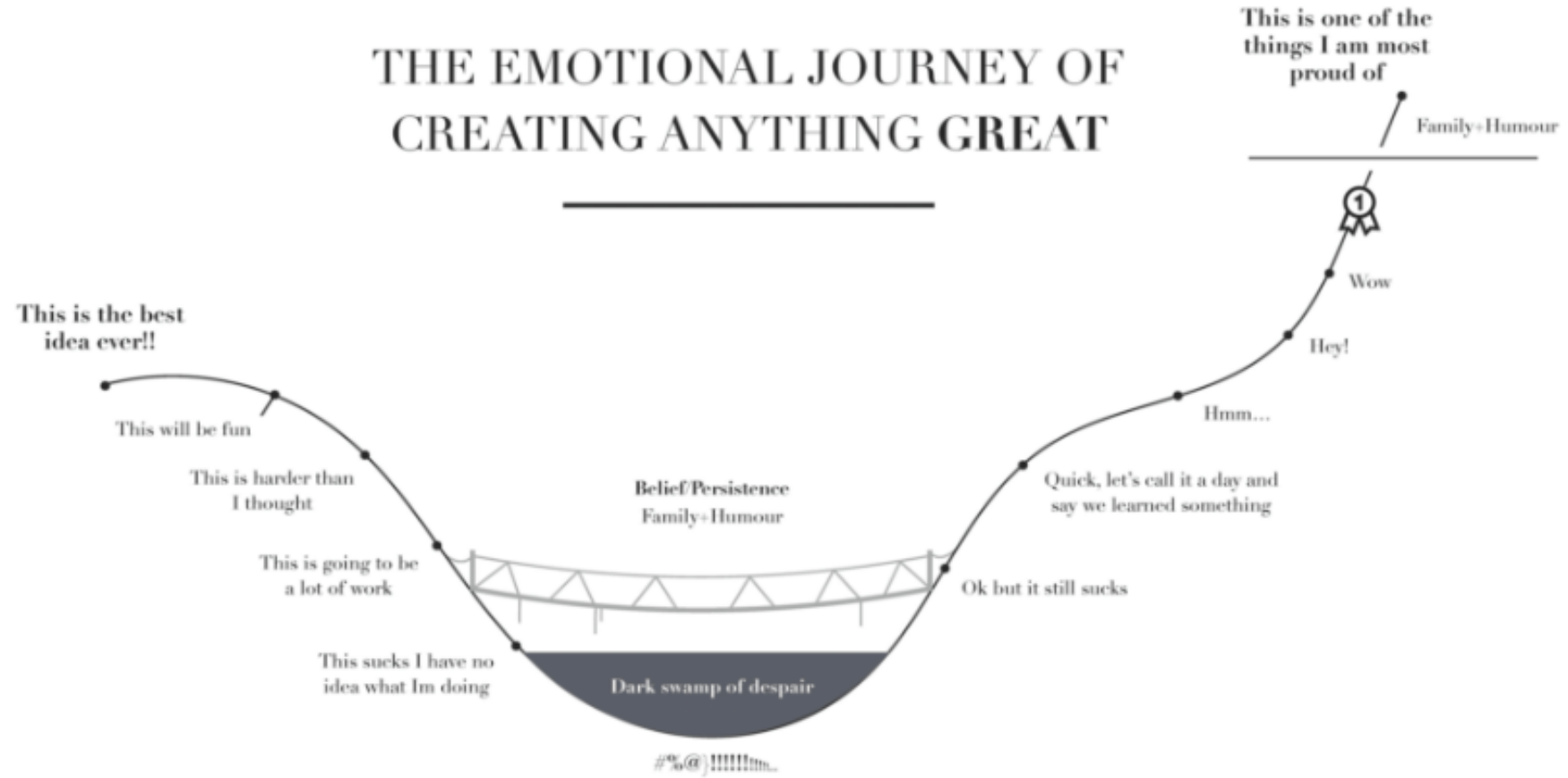


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Library Seminar Agenda

- Top 10 resources you should know about!
- What is neuroscience?
- Scholarly resources – books
- Scholarly resources – databases
- Scholarly resources – articles
- How to search for information
- Where, how and when to get help!
- Questions / Answers

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Library Seminar Agenda

- Top 10 resources you should know about!
- What is neuroscience?
- Scholarly resources – books
- Scholarly resources – databases
- Scholarly resources – articles
- How to search for information
- Where, how and when to get help!
- Questions / Answers

Top 10 resources you should know about!

1. Zoterobib
2. Zotero
3. Omni
4. Borrowing from Other Libraries
5. Brock Library NEUR Library Research Guide
6. Google Scholar, Google Books and Advanced Google searching
7. Open Education Resources (OERs)
8. PubMed / MEDLINE
9. When, where and how to get help!
10. Book a Consultation

Library Seminar Agenda

- Top 10 resources you should know about!
- [What is neuroscience?](#)
- Scholarly resources – books
- Scholarly resources – databases
- Scholarly resources – articles
- How to search for information
- Where, how and when to get help!
- Questions / Answers

What is neuroscience?

A word cloud of neuroscience-related terms. The words are arranged in a roughly circular pattern, with some terms being larger and more prominent than others. The colors of the words vary, including shades of green, blue, red, and purple. The background is a solid light gray.

chemistry
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mechanisms
neuroscience
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programming
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Neuroscience is the study of the nervous system and how it generates behaviour in animals and humans.

You'll study the link between neural function and behaviour at many levels by using techniques in molecular genetics, biochemistry, cellular physiology, neuroanatomy, electrophysiology, pharmacology, behavioural biology, neuropsychology, cognitive psychology and kinesiology. You'll also assist in active neuroscience laboratory research dealing with humans and non-humans.

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CHEMICALS / DRUGS

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Neuroscience

What is this guide for?

This guide has been designed as a general program guide and is curated by [Brock librarians](#). It features links to most often used resources such as databases for books, peer-reviewed journal articles, theses, dissertations, open educational resources (OEDs), standards, and more. Use the tabs on the left to navigate through the web page.

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Grey Literature

Grey literature [databases](#) identify resources that are key to research and scholarship. Many are open resources that go through a quality control process before they are published.

[Definitions](#) are scholarly dictionaries, encyclopedias and handbooks that help define terms and provide additional context.

[Concise Medical Dictionary](#) (2020) [Dictionary of Neurological Signs](#) (2011) [Oxford Companion to the Mind](#) (2004)

[Encyclopedia of Neuroscience](#) (2023)

[Cambridge Handbook of Intelligence and Cognitive Neuroscience](#) (2021)

[Gale Encyclopedia of Neurological Disorders](#) (2012)

[Netter's Atlas of Neuroscience](#) (2016)

[Taber's Cyclopedic Medical Dictionary](#) (2021)

[Oxford Handbook of Cognitive Neuroscience](#) (2013, vol 1 [Core Topics](#); vol 2 [The Cutting Edge](#))

Occupational Health and [Safety](#) information is essential to dealing with injuries, accidents, government and regulatory information.

[Theses and Dissertations](#) are important unpublished resources granted and retained by universities as capstone projects, these, and dissertations.

[Open Education Resources](#) (OERs) are digital ebooks and learning objects that can be used as open textbooks on a wide variety of general and disciplinary subjects.

[Tests and Measurements](#) are key assessment tools used to measure an individual's functioning and behavior to arrive at a diagnosis, treatment or conduct research. There are several key databases that describe individual tests and help with obtaining these resources.

Definitions

Defining terms is essential to scholarly research. Individual [Library Research Guides](#) may include alternate disciplinary encyclopedias, dictionaries, handbooks... and scholarly resources. The following databases may be a key resources to find key definitions. Individual reference resources can be identified using the [Omni](#) database.

[Google Dictionary](#) [Wiktionary](#) [Dictionary.com](#) [Cambridge Dictionary Online](#) [Merriam-Webster](#) [Word Hippo](#) (thesaurus)

Databases below are a select list of scholarly resources.

- **Oxford English Dictionary** [↗](#)

- Includes meaning, history, etymology, and pronunciation of words
- English language words from 1150 to the present
- **Permitted Uses**

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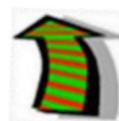
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neuroscience, *Noun*

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psychology,...

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neuroscience

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The Oxford Companion to the Body

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Subject: Science and technology, Life Sciences

Current Version: 2003

Length: 61 words

is the study of all aspects of nerves and the nervous system, in health and in disease. It includes the



neuroscience

Reference library

Oxford Dictionary of Biochemistry and Molecular Biology (2 ed.)

Reference type: **Subject Reference**

Subject: Science and technology, Life Sciences

Current Version: 2008

Length: 13 words

any of the sciences dealing with the nervous system; such sciences collectively.



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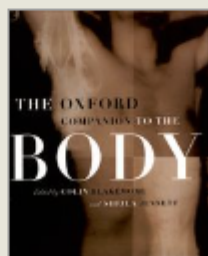
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Colin Blakemore and Sheila Jennett

Publisher: Oxford University Press

Print ISBN-13: 9780198524038

Current Online Version: 2003

Print Publication Date: 2001

Published online: 2003

eISBN: 9780191727511

neuroscience

is the study of all aspects of nerves and the nervous system, in health and in disease. It includes the anatomy, physiology, chemistry, pharmacology, and pathology of nerve cells; the behavioural and psychological features that depend on the function of the nervous system; and the clinical disciplines that deal with them, such as neurology, neurosurgery, and psychiatry.

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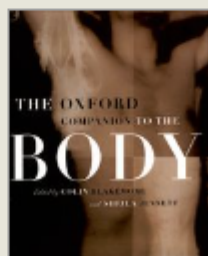
in *Oxford Dictionary of Biochemistry
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The Oxford Companion to the Body

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Current Online Version: 2003

Print Publication Date: 2001

Published online: 2003

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See [NERVOUS SYSTEM](#).



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Blakemore, C., & Jennett, S. (Eds.). (2001). *The Oxford companion to The body*.
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Exercise training and BDNF injections alter amyloid precursor protein (APP) processing e...	Baranowski et al.	2023-07-01
Routledge international handbook of social neuroendocrinology	Schultheiss et al.	2019
The Oxford companion to The body	Blakemore and Jennett	2001

Info Notes Tags Attachments Related

Item Type Book

Title The Oxford companion to The body

Editor Blakemore, Colin

Editor Jennett, Sheila

Series

Series Number

Volume

of Volumes

Edition

Place Oxford ; New York

Publisher Oxford University Press

Date 2001

of Pages 753

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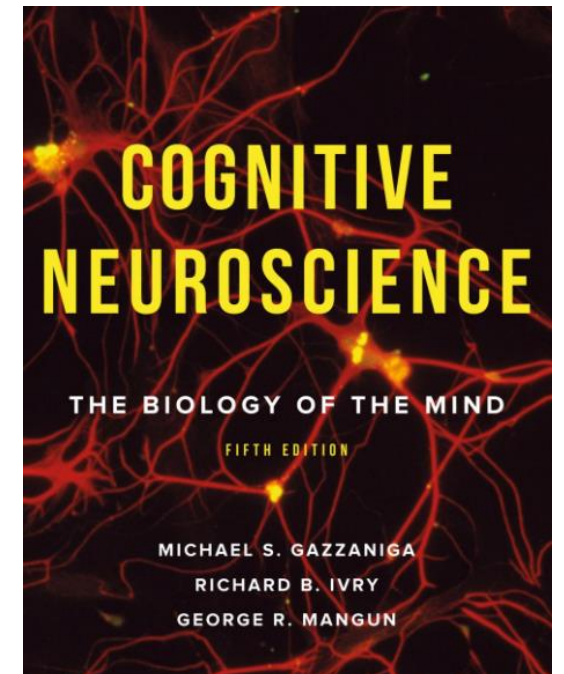
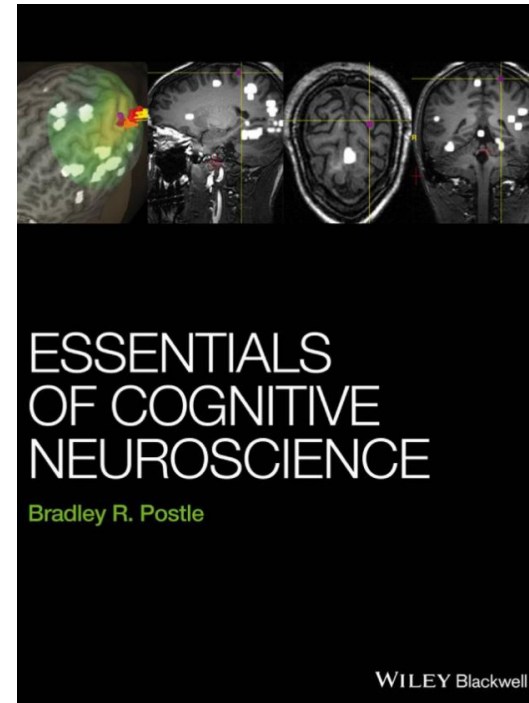
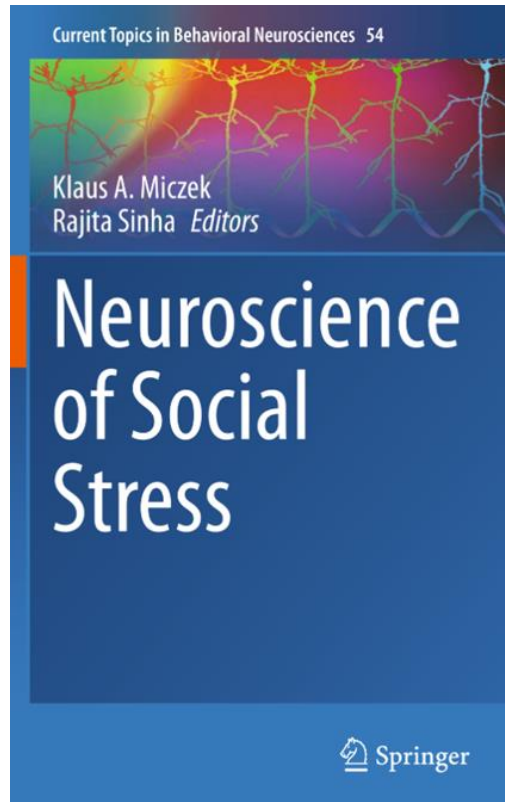
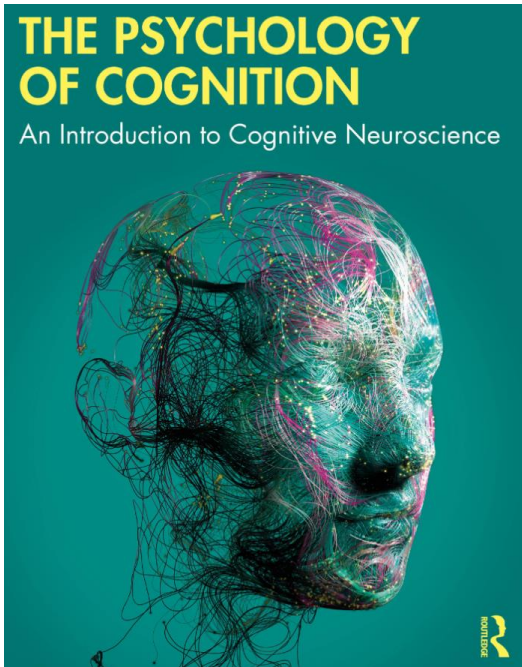
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Blakemore & Jennett (2001) define neuroscience as “...the study of all aspects of nerves and the nervous system...”

Blakemore, C., & Jennett, S. (2001). Neuroscience. In *The Oxford Companion to the Body*. Oxford.

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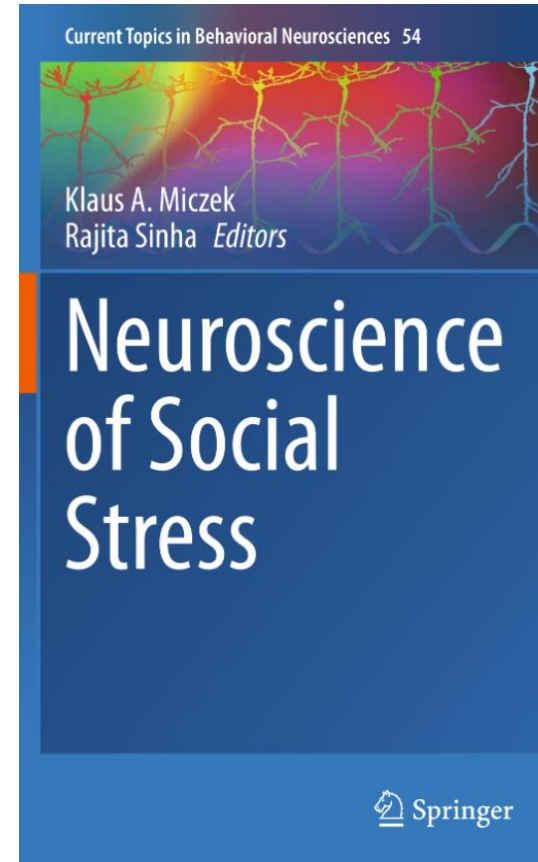
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Miczek, K. A., & Sinha, R. (Eds.). (2022).
Neuroscience of social stress. Springer.

ISBN: 3031042565 or 9783031042560

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Miczek, K. A., & Sinha, R. (Eds.). (2022). *Neuroscience of social stress* (Vol. 54). Springer International Publishing. <https://doi.org/10.1007/978-3-031-04256-0>

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10.1007/7854_2021_245

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McCormick, C. M. (2021). Methods and challenges in investigating sex-specific consequences of social stressors in adolescence in rats: Is it the stress or the social or the stage of development? In K. A. Miczek & R. Sinha (Eds.), *Neuroscience of Social Stress* (Vol. 54, pp. 23-58). Springer International Publishing.
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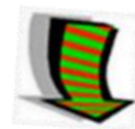
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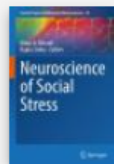
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Identifier	ISSN: 1866-3370 ISBN: 9783031042553 ISBN: 3031042557 EISSN: 1866-3389 EISBN: 9783031042560 EISBN: 3031042565 DOI: 10.1007/978-3-031-04256-0
Related titles	Current Topics in Behavioral Neurosciences
Publisher	Cham: Springer International Publishing

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Methods and Challenges in Investigating Sex-Specific Consequences of Social Stressors in Adolescence in Rats: Is It the Stress or the Social or the Stage of Development?



Cheryl M. McCormick

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Abstract Adolescence is a time of social learning and social restructuring that is accompanied by changes in both the hypothalamic-pituitary-gonadal axis and the hypothalamic-pituitary-adrenal (HPA) axis. The activation of these axes by puberty

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3 Sex Differences in Social Behaviour in Adolescence in Rats

3.1 Brain Regions Implicated in Social Behaviour

Social behaviour requires coordination among several brain regions that are reciprocally connected (see Fig. 1). The brain regions that are considered to be most critical to the network of social behaviour are the medial amygdala, bed nucleus of the stria terminalis, lateral septum, preoptic area; these brain regions have a high density of androgen and oestrogen receptors, as would be expected given the effects of sex hormones on, and sex differences in, social behaviour. These regions also are important sites of action of neuropeptides such as oxytocin and arginine vasopressin, which also have well-known roles in social behaviour (Dumais and Veenema 2016). Other regions such as the hippocampus, prefrontal cortex, nucleus accumbens, and paraventricular nucleus of the hypothalamus also contribute to social function. Many of these brain regions change significantly across the adolescent period. A review of the neural control of social behaviour and adolescent brain development is beyond the scope of this review. For reviews of these topics, see, Prounis and Ophir (2020), Bludau et al. (2019), Premachandran et al. (2020), and Shaw et al. (2020).

In this lab I observed that “...The brain regions that are considered to be most critical to the network of social behaviour are the medial amygdala...” (McCormick, 2001, 27).

McCormick, C. M. (2021). Methods and challenges in investigating sex-specific consequences of social stressors in adolescence in rats: Is it the stress or the social or the stage of development? In K. A. Miczek & R. Sinha (Eds.), *Neuroscience of Social Stress* (Vol. 54, pp. 23–58). Springer.
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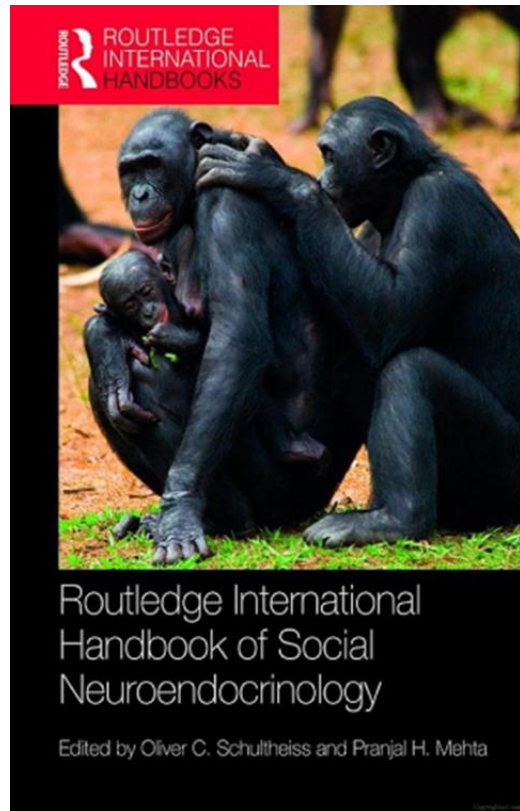
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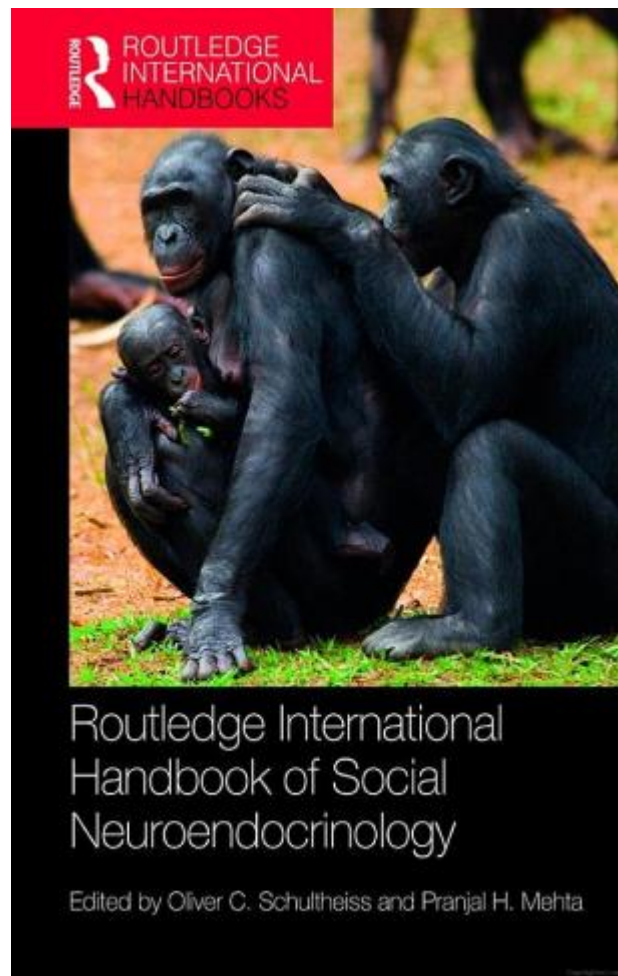
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STRESS AND SOCIAL DEVELOPMENT IN ADOLESCENCE IN A RODENT MODEL

Travis E. Hodges and Cheryl M. McCormick

Introduction

Neuroscientists (Cajal, 1967) and psychologists (Beach, 1950) have long recognized the importance of a comparative approach to understanding brain and behaviour relationships. The primary contribution of investigations in non-human animals for research in humans and vice versa is the generation of testable hypotheses and principles of development (Gottlieb & Lickliter, 2004). Nevertheless, research studies with animal models are increasingly conducted predominantly in rodents. Because of the increasing interest in the “social brain” in human adolescence and how stressors may influence its development, we review here, first, the typical social behaviour of adolescent laboratory rats, how social behaviour is measured and the development of the social brain in rats. Second, we review evidence from our lab of how social stress exposures (involving our social instability stress model) in adolescence influences social development. The majority of this research was conducted in males, and results described will be for males unless otherwise noted. We have reviewed the benefits and challenges of the use of rats for translational research on the consequences of stressors in adolescence (McCormick, Green, & Simone, 2017), and a greater discussion of the parallels and differences in adolescent development between the two species can be found in that review. For a review of other models of social stress used with adolescent rats, see Burke and colleagues (Burke, McCormick, Pellis, & Lukkes, 2017).

Adolescence, social behaviour and hypothalamic–pituitary–adrenal function in rats

Defining adolescence in rats

The specific ages in rats that define the adolescent period are variable across studies and across strains of rats. Although a liberal definition starts the adolescent period in rats at weaning from the mother (typically in the lab on postnatal day (PND) 21) and ends the adolescent period on PND 59 (with sexual maturity attained at about PND 60), this definition involves a lengthy prepubertal period. In contrast, in humans, adolescence is defined as beginning at puberty (reviewed in McCormick, Green, et al., 2017). In rats, the physical markers of puberty appear in

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STRESS AND SOCIAL DEVELOPMENT IN ADOLESCENCE IN A RODENT MODEL

Travis E. Hodges and Cheryl M. McCormick

Introduction

Neuroscientists (Cajal, 1967) and psychologists (Beach, 1950) have long recognized the importance of a comparative approach to understanding brain and behaviour relationships. The primary contribution of investigations in non-human animals for research in humans and vice versa is the generation of testable hypotheses and principles of development (Gottlieb & Lickliter, 2004). Nevertheless, research studies with animal models are increasingly conducted predominantly in rodents. Because of the increasing interest in the "social brain" in human adolescence and how stressors may influence its development, we review here, first, the typical social behaviour of adolescent laboratory rats, how social behaviour is measured and the development of the social brain in rats. Second, we review evidence from our lab of how social stress exposures (involving our social instability stress model) in adolescence influences social development. The majority of this research was conducted in males, and results described will be for males unless otherwise noted. We have reviewed the benefits and challenges of the use of rats for translational research on the consequences of stressors in adolescence (McCormick, Green, & Simone, 2017), and a greater discussion of the parallels and differences in adolescent development between the two species can be found in that review. For a review of other models of social stress used with adolescent rats, see Burke and colleagues (Burke, McCormick, Pellis, & Lukkes, 2017).

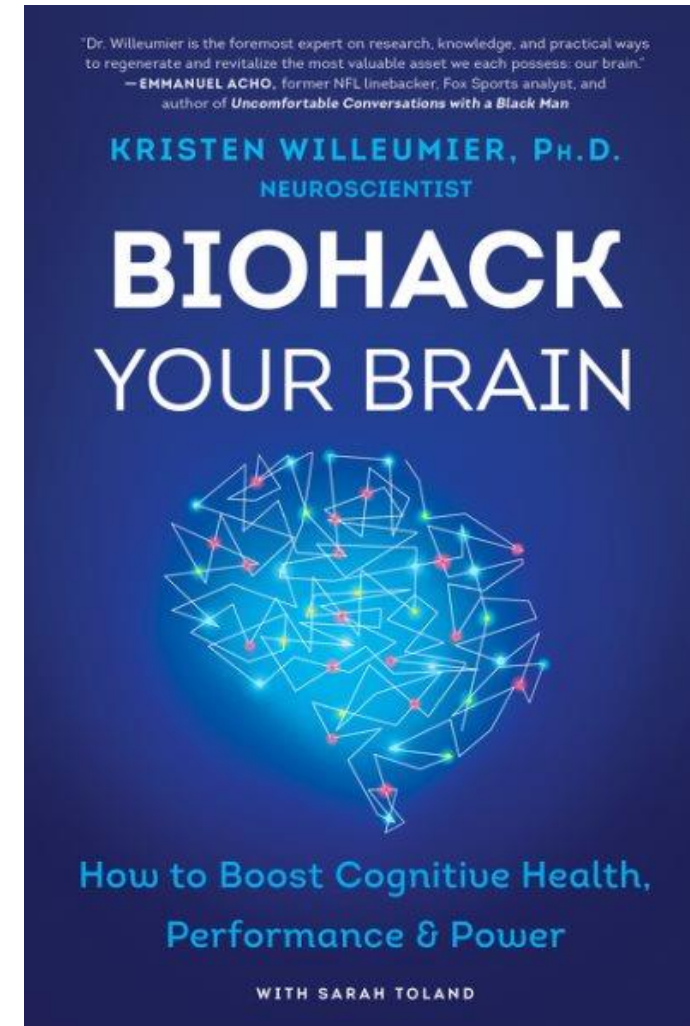
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In this paper I propose to define stress and social development in teenagers using previously discussed models (Hodges & McCormick, 2019)...

References

Hodges, T. E. & McCormick, C. M. (2019). Stress and social development in adolescence in a rodent model. In, O. C. Schultheiss & P. H. Mehta (Eds.), Routledge international handbook of social neuroendocrinology (pp. 479-503). Routledge.

Defining 'Cool Climate' Research: A Bibliographic Analysis

Ian D. Gordon¹, Betty Galbraith², Chris Savino³

¹ James A. Gibson Library, Brock University, St. Catharines, ON, L2S 3A1, Canada.

² Owen Science Library, WSU Libraries, Washington State University, Pullman WA, 99164-5610, United States.

³ College of Agriculture and Life Sciences, Cornell University, Ithaca NY, 14853, United States.



Introduction

What counts as "cool climate" research continues to be open for debate by oenologist, viticulturalists, researchers, scholars, practitioners and wine lovers (Gardner 2016, Mowery 2016, Puckette 2018). Conducting a bibliographic analysis of cool climate scholarly papers and their journals, this study helps to provide data and more clarity as to what constitutes "cool climate" wine research. Bibliometric studies provide a critical quantitative evaluation of publishing, subject, and scholarly impact. This bibliometric study of "cool climate" wine scholarly papers and their journals is truly unique in its analysis and findings.

Gardner, Dennis. "What is Cool?" (2016) Paper Presented at the 2016 International Cool Climate Wine Symposium, St. Catharines, Ontario, Canada, July 17-21, 2016.

Mowery, Lauren. (2016). The Best of Both Worlds: Cool Climate and Warm Climate Wine. Washington State University.

Methodology

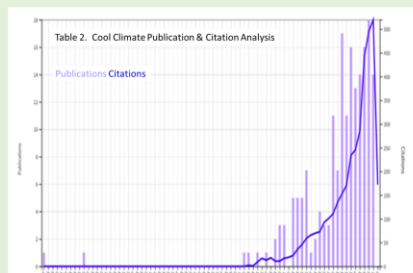
Three datasets were created using: 1) Clarivate Web of Science Complete and Elsevier Scopus database bibliographic search strategies ["cool climate" and wine*] AND (grape* or enology* or oenology* or viticulture* or viti*) AND P(=2014-2018); 2) Clarivate InCites and Elsevier SciVal database benchmarking topic clusters and research categories e.g. TC, S44, and 3) published papers by researchers at or connected with Brock's CCOVI Institute, Cornell and WSU's Viticulture and Enology programs from 2014 to 2018. Data visualizations, benchmarking and statistical analysis were not limited to but included metrics from Journal Citation Reports (JCR), Altmetric Explorer, Scimago SJR, InCites, SciVal, Google Scholar, Harzing.com Publish or Perish, Bibliometrix R Package, CWTs Journal indicators and additional alternate databases.



Findings

- Included 562 aggregated papers from 232 journals by **Brock (17.6%), Cornell (50.5%), and WSU (33.5%)** researchers
- Analyzed **7012 "cool climate" papers** in total, from **1127 journals**
- **Top 25 cool climate journals and their impact values**, see **Table 1**
- Cool climate research is **expanding exponentially**, 19.63 average citations per item, 30 h-index, see **Table 2**
- A significant **majority (79.9%) of "cool climate" papers** came from a **minority (21.2%) of academic journals**
- **Cool climate research is respectable**, 5-year aggregated mean cool climate **journal impact factor (2.681)** is respectable wrt Horticulture (1.597), Food Science & Technology (2.851), Biology (3.181), Ecology (3.385), Plant Sciences (4.414)...
- **Top 50 (of 96) cool climate subject clusters**, Food Science (9.6%), Agronomy (8.3%), Plant Science (7.1%), Horticulture (4.9%), Insect Science (3.4), and Ecology (2.9%), see **Figure 1**
- **Top cool climate indexing and abstracting databases**: Scopus (88.4%), Web of Science (85.9%), BIOSIS (70.3%), Vitis (51.2%), SciFinder-n (49.8), AGRIS (46.3%), MEDLINE (44.5%), FSTA (36.3%), AGRICOLA (35.8%)...
- **Top cool climate researches co/published on average 3.21 papers per year**, WSU (2.35), Brock (3.96), and Cornell (4.04)
- 2.7% of viticulture and oenology scholarly articles are tagged with **"cool climate" descriptors**
- 24.3% of cool climate papers were published in **open access journals**
- 3.8% of papers involve **non-scientific** research areas.

Journal Title	Impact
1 Australian Journal of Grape and Wine Research	21.40
2 Food Chemistry	24.72
3 Science Direct	24.80
4 Journal of Agricultural and Food Chemistry	20.08
5 American Journal of Enology and Viticulture	22.00
6 Food Science and Technology	22.00
7 VIT - Food Science and Technology	24.40
8 Journal of Food Science	24.00
9 Journal of the Science of Food and Agriculture	22.00
10 Food Chemistry	22.00
11 South African Journal of Enology and Viticulture	20.00
12 Food Research International	20.00
13 Molecules	9.36
14 Academy of Management Journal	1.24
15 Journal International des Sciences de la Vigne et du Vin	4.90
16 HortScience	1.64
17 Journal of Applied Microbiology	1.76
18 Journal Review of Enology	1.20
19 Journal of Economic Entomology	1.20
20 HortTechnology	1.24
21 European Journal of Agronomy	4.05
22 1993 - Journal of Enology Research	4.17
23 European Food Research and Technology	4.70
24 International Journal of Food Microbiology	4.88
25 HortTechnology	4.48



Cool climate wine research is interdisciplinary, international, prospering, and an important scientific discipline. Bibliometrics can help quantify research impact and outcomes. Academic librarians can support researchers with evidence synthesis, bibliometrics, graphics, visual presentations of data and assist with telling great stories.

Gordon, I. D., Galbraith, B., & Savino, C. (2022, July). *Defining cool climate Research: A bibliographic analysis*. [Poster Presentation]. International Cool Climate Wine Symposium, Brock University. <https://archive.org/details/gordon-galbraith-savino-defining-cool-climate-research-a-bibliographic-analysis->



Defining Cool Climate Research: A Bibliographic Analysis

by Ian D. Gordon



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Poster presented at the International Cool Climate Symposium held at Brock University, St. Catharines, Ontario, Canada, July 17-21, 2022. Poster created and presented by Ian D. Gordon, Brock University, Betty Galbraith, Washington State University, and Chris Savino, Cornell University.

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[Gale Encyclopedia of Neurological Disorders](#) (2012)

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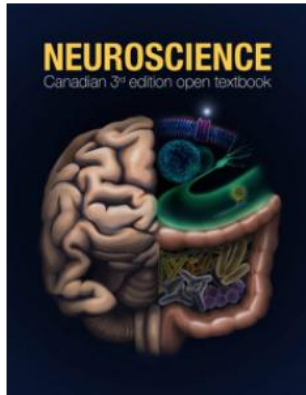
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5.6 Integrative and Contemplative Neuroscience

Sunny Sanghee Baek and Bushra Ahmed

About the Author/Contributors

How is Music Processed?

It is important to learn how our brains process music to be able to implement music therapies and make informed choices for various brain traumas and neurological disorders. There is an inherent difficulty in the understanding of music processing due to the sheer plethora of music genres and the different neurological cascades they can kick-start by evoking different emotions such as stress, joy, happiness, melancholy, et cetera (Hernandez-Ruiz, 2019; Tai & Kuo, 2019). Each musical melody is made up of different elements which can be randomly categorized under pitch, tempo and rhythm, volume, timbre, texture, duration, and form. Out of these, only pitch and tempo and rhythm have been studied in detail (Hernandez-Ruiz, 2019).

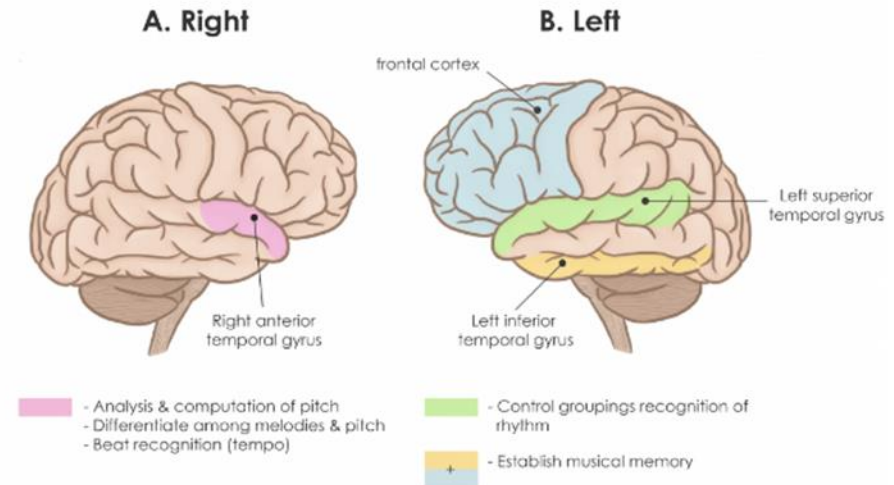


Figure 1. Different functions of the left and right hemispheres in processing music. **A.** Right hemisphere **B.** Left hemisphere.

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Body

Adults and children with cognitive disorders may have trouble walking – but neurologic music therapy can be a helpful part of their treatment plan, according to growing research.

Neurologic music therapy has been shown to help retrain the brain to walk, improving the gait of those with [multiple sclerosis](#), Parkinson's, stroke and cerebral palsy.

A recent small pilot [study](#) adds to that growing body of evidence, with preliminary findings that show the therapy could benefit those with brain injuries as well. The children, who were brain injury patients with trouble walking, showed improvements in speed, posture and steadiness.

"There's something very fundamental and substantial happening in the brain when we listen to music, especially when we try to use this for improving mobility," said Michael Thaut, a University of Toronto professor of music and rehabilitation science who directs the Music and Health Science Research Center. "Our natural response to music is to move."

What is neurologic music therapy and how does it work?

This therapy is used in rehabilitation and physiotherapy settings and can help patients with language, speech, sensory and motor difficulties, according to the [Brain Injury Association of America](#).

Different strategies are used for different conditions. One neurologic music therapy strategy used to help patients with walking, balance and coordination is called rhythmic auditory stimulation (RAS). This was used with the four children in the study published in the International Journal of Therapy and Rehabilitation, which included the Cambridge Institute for Music Therapy Research in the United Kingdom.

RAS uses sound to cue brain signals to take steps and keep up with a rhythm. A trained therapist uses music or a metronome to help guide a patient, often on a treadmill.

The patient walks in synchronicity to the beat with a goal to maintain the speed and rhythm "without the auditory stimulus," Thaut explained.

"It's a therapy regimen that goes for days, every day," he said. "That sense of regularity translates into a more regular stepping pattern."

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Exercise training and BDNF injections alter amyloid precursor protein (APP) processing enzymes and improve cognition

Bradley J. Baranowski , Ahmad Mohammad, Michael S. Finch , Alex Brown, Roopan Dhaliwal, Daniel M. Marko , Paul J. LeBlanc, Cheryl M. McCormick, Val A. Fajardo , and Rebecca E. K. MacPherson  [Show fewer authors](#) 

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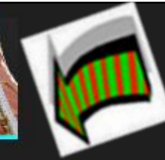
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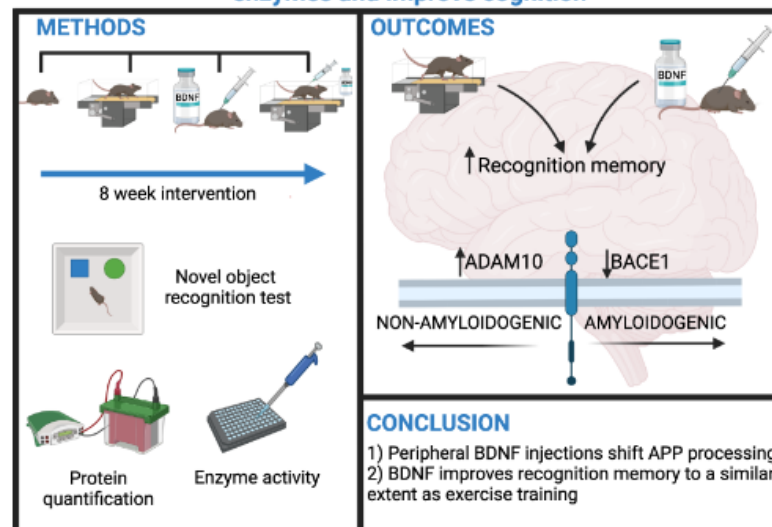
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Exercise training and BDNF injections alter APP processing enzymes and improve cognition



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Physical Activity and the Brain

Exercise training and BDNF injections alter amyloid precursor protein (APP) processing enzymes and improve cognition

Bradley J. Baranowski,¹ Ahmad Mohammad,¹ Michael S. Finch,¹ Alex Brown,¹ Roopan Dhaliwal,¹
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Abstract

Exercise reduces cognitive aging, neurodegeneration, and Alzheimer's disease (AD) risk. Acute exercise reduces the activity of β -site amyloid precursor protein-cleaving enzyme 1 (BACE1), the rate-limiting enzyme in the production of A β . However, mechanisms mediating these effects remain largely unknown. Work has implicated brain-derived neurotrophic factor (BDNF) in the processing of amyloid precursor protein (APP). BDNF is an exercise-induced neurotrophin known for its role in synaptic plasticity, neurite growth, and neuronal survival. Previously, our lab has shown using an ex vivo model that treatment with BDNF reduced BACE1 activity, highlighting a BDNF to BACE1 link. The purpose of this research was to determine if BDNF treatments resulted in similar biochemical adaptations to APP processing as exercise training. Male mice were assigned into one of four groups ($n = 12$ /group): 1) control; 2) exercise training (progressive treadmill training (0.5 mg/kg body mass subcutaneous injection 5 days/wk); or 4) endurance training and BDNF, for a 4-week period. Recognition memory was measured with a novel object recognition test. Serum, the prefrontal cortex, and hippocampus were collected. BDNF improved recognition memory to a similar extent as endurance training. BDNF and exercise training increased ADAM10 activity in the prefrontal cortex, indicating a shift in APP processing. Our findings suggest that BDNF exerts similar beneficial effects on cognition and APP processing as exercise training. Future evidence-based preventative or therapeutic interventions that increase BDNF and reduce BACE1 will be of value for populations that are at risk of AD.

NEW & NOTEWORTHY Our study presents the novel findings that chronic peripheral BDNF injections result in similar biochemical adaptations to APP processing enzymes and improved cognition to a similar extent as exercise training. These findings highlight the potential efficacy of using BDNF as a therapeutic intervention in the prevention of neurodegenerative diseases (i.e., Alzheimer's disease). Furthermore, future evidence-based preventative or therapeutic interventions that increase BDNF and reduce BACE1 will be of value for populations that are at risk of AD.

amyloid precursor protein; BDNF; brain; cognition; exercise training

INTRODUCTION

The overproduction and accumulation of amyloid beta (A β) peptides that can result in plaque formation is a key feature of neurodegenerative diseases, such as Alzheimer's disease (AD). A β peptides have been shown to lead to neuronal loss, cognitive impairments, neuroinflammation, synaptic dysfunction, and other neurotoxic events in the brain (1). Alternatively, a lifelong underproduction of A β , as observed in individuals with the Icelandic mutation in amyloid precursor protein (APP), protects individuals from cognitive decline with aging (2). Increased production of these peptides can be

observed decades before clinical diagnosis (3–6), highlighting the importance of early, preventative measures to reduce the A β burden as one ages. A β originates from a large transmembrane protein, known as the APP (7, 8). The fate of APP processing is determined by one of two competing pathways: the amyloidogenic and nonamyloidogenic pathways (9). The amyloidogenic pathway is initiated when the rate-limiting enzyme, β -site amyloid precursor protein cleaving enzyme 1 (BACE1), cleaves APP resulting in the release of a soluble ectodomain, soluble APP β (sAPP β) fragment, and leaving behind a membrane-bound C-terminal fragment (CTF) 89/99 (10, 11). γ -Secretase then cleaves the CTF89/99 fragment and releases

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INTRODUCTION

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ing was effective in increasing resting peripheral BDNF levels (43). In humans, BDNF appears to increase in intensity- (42) and duration-dependent manner (43). A similar intensity-dependent response is seen within rodent models (44). Specifically, Soya et al. compared low- and moderate-intensity treadmill running in a rat model (30 min at 15 m/min vs. 25 m/min). This study demonstrated that a single bout of exercise significantly increases BDNF mRNA expression in the hippocampus, with levels peaking at 1.5 h postexercise (44). BDNF appears to increase with alternative modalities of exercise (i.e., strength training), however, these effects appear more transient than endurance training (45). The exact mechanism for exercise-induced increases in BDNF expression remains contentious in nature. Elevated levels of BDNF

METHODS AND MATERIALS

Animals and Ethics

Male 10-wk-old C57BL6/J mice were purchased from The Jackson Laboratory (Bar Harbor, ME). Mice acclimatized for 7 days in the Brock University Comparative Biosciences Facility. All mice were kept on a 12-h light:dark cycle and had ad libitum access to food and water through the entirety of the study. Experimental protocols were approved by the Brock University Animal Care Committee (AUP: #20-07-04) and are in compliance with the Canadian Council on Animal Care.

Experimental Design

Male C57BL6/J mice were randomized into one of four groups ($n = 12/\text{group}$): 1) control (CON); 2) exercise (EX); 3) BDNF; or 4) exercise and BDNF (EX + BDNF). Baseline measures of all mice ($n = 48$) were conducted immediately after acclimatization but before group assignment to ensure no initial differences in body mass. The study was conducted over an 8-wk period and body mass (g) and food intake (g/wk) were recorded weekly. During the last week of the intervention, Promethion behavioral phenotyping was recorded for 24 h, and object recognition testing was performed.

Treadmill Training Protocol

Mice in the EX groups performed treadmill running for 1

of 60 min. Mice were then placed back in their respective boxes with one familiar object and one novel object, exploring for 10 min. Exploration index (or percent of total investigation time) was calculated as the percent of the time spent investigating the novel object divided by the sum of the times spent with both objects (56). This protocol was repeated the following 2 days with varying ITIs (2 h and 4 h, respectively), different familiar and novel objects were used with each ITI testing day.

Behavioral Phenotyping

A subset of mice ($n = 8/\text{group}$) was housed in Promethion high-definition behavioral phenotyping cages (Sable Systems, North Las Vegas, NV) for 24 h. This system allows for real-time behavior analysis of mice in a home

Cell Signaling, Cat. No. 6986S), BACE1 (1:500, Cell Signaling, Cat. No. 5606P), pBACE1 Thr 252(1:1,000, Affinity, Cat. No. AF3650), pBACE1 Ser498 (1:1,000, Invitrogen, Cat. No. PA5-118602), Akt (1:1,000; Cell Signaling, Cat. No. 4685S), pAkt Ser473 (1:1,000; Cell Signaling, Cat. No. 4058S), GSK3 β (1:1,000; Cell Signaling, Cat. No. 9315S), pGSK3 β Ser 9 (1:1,000, Cell Signaling, Cat. No. 5558S), and ADAM10 (1:1,000, Abcam, Cat. No. 1997) were obtained and used during this study.

Enzyme Activity

BACE1.

BACE activity in the prefrontal cortex and hippocampus was determined using a commercially available β -secretase activity assay kit (Ab65357) as previously described (27, 59). Samples were homogenized (FastPrep, MP Biomedicals, Santa Ana, CA) and extracted using 20 volumes of ice-cold PBS. Samples were left to incubate on ice for 15 min and centrifuged at 10,000 *g* for 5 min at 4°C. The supernatant was collected, and a BCA assay was performed to determine protein concentration. All samples were prepared at 0.50 μ g/ μ L. A total of 50 μ L of sample was added to each well in duplicate, followed by 50 μ L of 2 $\times$ reaction buffer and 2 μ L of β -secretase substrate. The plate was left to incubate in the dark at 37°C for 60 min and fluorescence was read using a spectrometer (SpectraMax M2; Molecular Devices) at excitation and emission wavelengths of 335 and 495 nm, respectively.

ADAM10.

ADAM10 activity in the prefrontal cortex and hippocampus was determined using a commercially available ADAM10 activity assay kit (Anaspec; AS-72226). Samples were homogenized and prepared as described in the BACE1 activity assay. A total of 50 μ L of sample was added to each well in duplicate, followed by 50 μ L of the unique FRET (5-FAM)

RESULTS

Changes in Whole Body Measurements and Cognitive Behavior

There were no differences in body mass between the groups of mice ($P > 0.05$) before the initiation of the study. After 8 wk of intervention, BDNF-treated mice had a lower body mass than saline-injected mice (Fig. 1A; $P = 0.0001$). BDNF treatment reduced daily food intake (Fig. 1B; $P = 0.0006$). BDNF injections and exercise training had higher serum concentrations of BDNF than CON (Fig. 1C; $P = 0.01$). Promethion behavioral phenotyping demonstrated that BDNF and exercise did not change mouse cage behavior, indicated by no differences in short lounge time, habitat interaction, water intake, water interaction, food intake, and food interaction (Fig. 1D). However, BDNF mice spent more time in their habitats and had shorter long lounge times (indicated by no sensor interaction for 60 s) compared with CON mice (Fig. 1D).

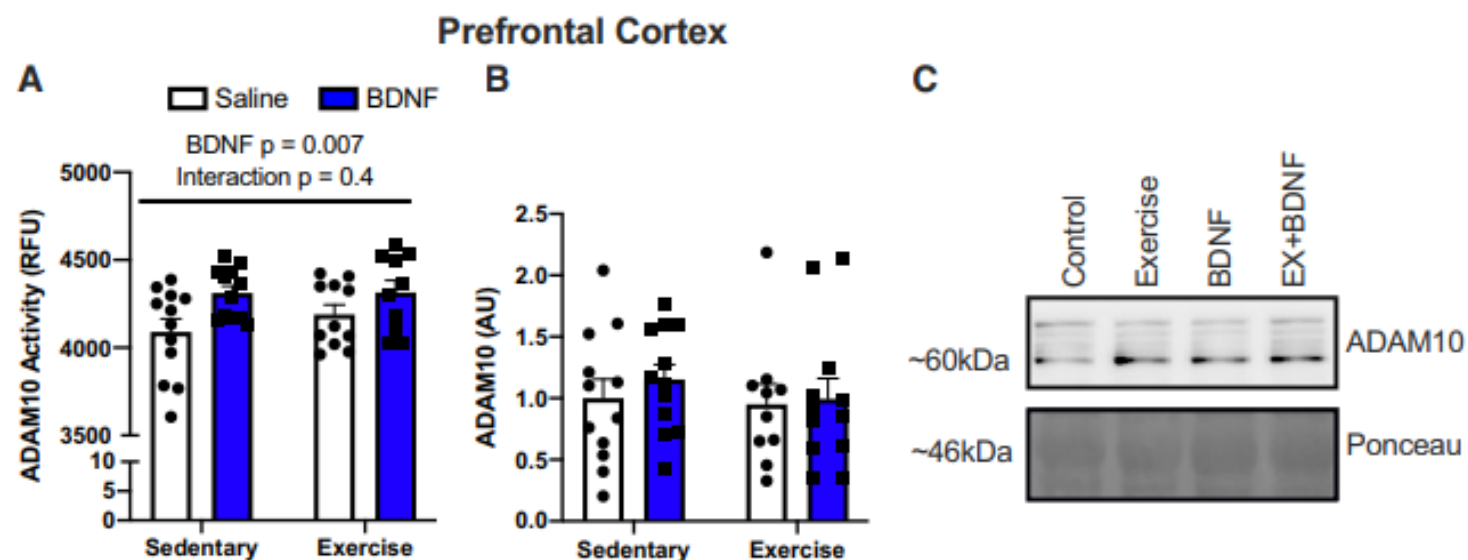
BDNF and Endurance Training Promote Novel Object Recognition

To determine the effects of exercise and BDNF on cognitive function, a NORT was used to assess recognition memory. A three-way ANOVA indicated a main effect for BDNF treatment (Fig. 2A; $P = 0.01$), exercise training ($P = 0.03$), and ITI ($P = 0.03$), however, there were no interactions ($P = 0.6$). BDNF-injected and exercise-trained mice had a higher percent exploration of the novel object compared with the familiar control object than CON mice, whereas percent exploration of the novel object was reduced with subsequent ITI regardless of the intervention group. Effect size at the 1-h ITI was BDNF ($d = 0.32$), EX ($d = 0.18$), EX + BDNF ($d =$

Changes in BDNF Signaling

In the prefrontal cortex, there was an interaction with BDNF injections within the sedentary groups, and post hoc analysis demonstrated higher mature BDNF (mBDNF) content in the BDNF mice than in CON mice (Fig. 5A; $P = 0.007$; $d = 0.79$). There was a main effect of exercise training to

–0.49). Although there were no differences in pro-BDNF content across groups ($P = 0.13$), there was a main effect of BDNF injections, which resulted in a lower ratio of mature/pro-BDNF (Fig. 5C; $P = 0.04$; $d = -0.63$). In the hippocampus, there was an interaction with BDNF injections within the exercise group, and post hoc analysis revealed that the EX + BDNF group had higher total TrkB content than



DISCUSSION

This study provides a novel examination of the relationship between chronic BDNF treatment, exercise training, and the subsequent effects on APP processing enzymes and cognitive behavior. This is the first study to demonstrate that in an in vivo model, chronic peripheral BDNF injections resulted in a shift in APP processing toward the nonamyloidogenic pathway and improved recognition memory, similar to endurance training. Our results further highlight regional differences (prefrontal cortex vs. hippocampus) in response to endurance training and BDNF treatment. Specifically, we demonstrate that endurance training and BDNF reduce BACE1 activity to a similar extent in the prefrontal cortex, but no reduction was observed in the hippocampus. Furthermore, our results demonstrate that BDNF alone not only alters BACE1 activity but also increases ADAM10 activity in both the prefrontal cortex and hippocampus. This work highlights the potential of BDNF to alter APP through

Conclusions

This study provides novel findings regarding the regulation of exercise and BDNF on APP processing. Our data show that both exercise training and chronic BDNF injections can modulate BACE1 activity in a brain region-specific manner, however, BDNF injections alone were able to elevate the activity of ADAM10, phosphorylate BACE1 Ser498 (presumably increase retrograde trafficking), and elevate GSK3 β inhibition via Akt signaling. These findings demonstrate that exercise regulates APP processing differently than BDNF alone and that BDNF appears to have a more potent effect on APP processing. This may indicate that direct treatment with BDNF provides a more effective therapeutic strategy for modulating APP in a beneficial manner. We have also demonstrated that

DATA AVAILABILITY

Data will be made available upon reasonable request.

ACKNOWLEDGMENTS

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GRANTS

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

B.J.B., P.J.L., C.M.M., V.A.F., and R.E.K.M. conceived and designed research; B.J.B., A.M., M.S.F., A.B., R.D., D.M.M., and R.E.K.M. performed experiments; B.J.B. analyzed data; B.J.B. interpreted results of experiments; B.J.B. prepared figures; B.J.B. and R.E.K.M. drafted manuscript; B.J.B., A.M., M.S.F., A.B., R.D., D.M.M., P.J.L., C.M.M., V.A.F., and R.E.K.M. edited and revised manuscript; B.J.B., A.M., M.S.F., A.B., R.D., D.M.M., P.J.L., C.M.M., V.A.F., and R.E.K.M. approved final version of manuscript.

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Exercise training and BDNF injections alter amyloid precursor protein (APP) processing enzymes and improve cognition

By

Baranowski, BJ (Baranowski, Bradley J.) ^[1]; Mohammad, A (Mohammad, Ahmad) ^[1]; Finch, MS (Finch, Michael S.) ^[1]; Brown, A (Brown, Alex) ^[1]; Dhaliwal, R (Dhaliwal, Roopan) ^[1]; Marko, DM (Marko, Daniel M.) ^[1]; LeBlanc, PJ (LeBlanc, Paul J.) ^[1]; McCormick, CM (McCormick, Cheryl M.) ^[2], ^[4]; Fajardo, VA (Fajardo, Val A.) ^[3], ^[4]; MacPherson, REK (MacPherson, Rebecca E. K.) ^[1], ^[4]

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Abstract

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Keywords

Author Keywords: amyloid precursor protein; BDNF; brain; cognition; exercise training

Keywords Plus: NEUROTROPHIC FACTOR LEVELS; ALZHEIMERS-DISEASE; PHYSICAL-ACTIVITY; BLOOD-BRAIN; MOUSE MODEL; PRECLINICAL STAGES; AEROBIC EXERCISE; BETA; PHOSPHORYLATION; IMPAIRMENT

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Endocannabinoid system contributions to sex-specific adolescent neurodevelopment

Jonathan J Simone¹, Matthew R Green², Cheryl M McCormick³

Affiliations  expand
PMID: 34534603 DOI: 10.1016/j.pnpbp.2021.110438

Abstract

With an increasing number of countries and states adopting legislation permitting the use of cannabis for medical purposes, there is a growing interest among health and research professionals into the system through which cannabinoids principally act, the endocannabinoid system (ECS). Much of the seminal research into the ECS dates back only 30 years and, although there has been tremendous development within the field during this time, many questions remain. More recently, investigations

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With an increasing number of countries and states adopting legal cannabis, there is a growing interest among health and research professionals in the endocannabinoid system (ECS). Much of the seminal research on the ECS has been tremendous development within the field during this time. Recently, it has emerged examining the contributions of the ECS to normative adolescent development and critical periods. One such period is adolescence, a unique period of brain development in preparation for adulthood, including shifts in endocannabinoid signaling and regarding the maturation of the ECS during adolescence and

Keywords

Endocannabinoid; CB1 receptor; Adolescence; Neurodevelopment; Behaviour; Intracellular signalling

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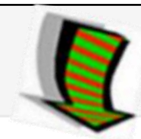


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RESEARCH ARTICLE

Physical Activity and the Brain

Exercise training and BDNF injections alter amyloid precursor protein (APP) processing enzymes and improve cognition

Bradley J. Baranowski,¹ Ahmad Mohammad,¹ Michael S. Finch,¹ Alex Brown,¹ Roopan Dhaliwal,¹ Daniel M. Marko,¹ Paul J. LeBlanc,¹ Cheryl M. McCormick,^{2,4} Val A. Fajardo,^{3,4} and Rebecca E. K. MacPherson^{1,4}

¹Department of Health Sciences, Brock University, St. Catharines, Ontario, Canada; ²Department of Psychology, Brock University, St. Catharines, Ontario, Canada; ³Department of Kinesiology, Brock University, St. Catharines, Ontario, Canada; and ⁴Centre for Neuroscience, Brock University, St. Catharines, Ontario, Canada

Abstract

Exercise reduces cognitive aging, neurodegeneration, and Alzheimer's disease (AD) risk. Acute exercise reduces the activity of β -site amyloid precursor protein-cleaving enzyme 1 (BACE1), the rate-limiting enzyme in the production of A β . However, mechanisms mediating these effects remain largely unknown. Work has implicated brain-derived neurotrophic factor (BDNF) in the processing of amyloid precursor protein (APP). BDNF is an exercise-induced neurotrophin known for its role in synaptic plasticity, neurite growth, and neuronal survival. Previously, our lab has shown using an ex vivo model that treatment of the prefrontal cortex with BDNF reduced BACE1 activity, highlighting a BDNF to BACE1 link. The purpose of this research was to examine whether BDNF treatments resulted in similar biochemical adaptations to APP processing as exercise training. Male C57BL/6J mice were assigned into one of four groups ($n = 12$ /group): 1) control, 2) exercise training (progressive treadmill training 5 days/wk); 3) BDNF (0.5 mg/kg body mass subcutaneous injection 5 days/wk); or 4) endurance training and BDNF, for an 8-wk intervention. Recognition memory was measured with a novel object recognition test. Serum, the prefrontal cortex, and hippocampus were collected. BDNF improved recognition memory to a similar extent as endurance training. BDNF and exercise decreased BACE1 activity and increased ADAM10 activity in the prefrontal cortex, indicating a shift in APP processing. Our novel results indicate that BDNF exerts similar beneficial effects on cognition and APP processing as exercise training. Future evidence-based preventative or therapeutic interventions that increase BDNF and reduce BACE1 will be of value for populations that are at risk of AD.

NEW & NOTEWORTHY Our study presents the novel findings that chronic peripheral BDNF injections result in regulation of APP processing enzymes and improved cognition to a similar extent as exercise training. These findings highlight the potential efficacy of using BDNF as a therapeutic intervention in the prevention of neurodegenerative diseases (i.e., Alzheimer's disease). Furthermore, future evidence-based preventative or therapeutic interventions that increase BDNF and reduce BACE1 will be of value for populations that are at risk of AD.

amyloid precursor protein; BDNF; brain; cognition; exercise training

INTRODUCTION

The overproduction and accumulation of amyloid beta (A β) peptides that can result in plaque formation is a key feature of neurodegenerative diseases, such as Alzheimer's disease (AD). A β peptides have been shown to lead to neuronal loss, cognitive impairments, neuroinflammation, synaptic dysfunction, and other neurotoxic events in the brain (1). Alternatively, a lifelong underproduction of A β , as observed in individuals with the Icelandic mutation in amyloid precursor protein (APP), protects individuals from cognitive decline with aging (2). Increased production of these peptides can be

observed decades before clinical diagnosis (3–6), highlighting the importance of early, preventative measures to reduce the A β burden as one ages. A β originates from a large transmembrane protein, known as the APP (7, 8). The fate of APP processing is determined by one of two competing pathways: the amyloidogenic and nonamyloidogenic pathways (9). The amyloidogenic pathway is initiated when the rate-limiting enzyme, β -site amyloid precursor protein-cleaving enzyme 1 (BACE1), cleaves APP resulting in the release of a soluble ectodomain, soluble APP β (sAPP β) fragment, and leaving behind a membrane-bound C-terminal fragment (CTF) 89/99 (10, 11). γ -Secretase then cleaves the CTF89/99 fragment and releases

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My investigation attempts to find literature that helps to explain “...if BDNF treatment alone results in similar changes to APP processing enzymes as endurance training and if any observed biochemical molecular changes are accompanied by behavioral/functional improvements” (Baranowski et al, 2023, 122).

Baranowski, B. J., Mohammad, A., Finch, M. S., Brown, A., Dhaliwal, R., Marko, D. M., LeBlanc, P. J., McCormick, C. M., Fajardo, V. A., & MacPherson, R. E. K. (2023). Exercise training and BDNF injections alter amyloid precursor protein (App) processing enzymes and improve cognition. *Journal of Applied Physiology*, 135(1), 121–135.
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What are the main concepts?

...if BDNF treatment alone results in similar changes to APP processing enzymes as endurance training and if any observed biochemical molecular changes are accompanied by behavioral/functional improvements.

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What are the main concepts?

...if BDNF treatment alone results in similar changes to APP processing enzymes as endurance training and if any observed biochemical molecular changes are accompanied by behavioral/functional improvements.

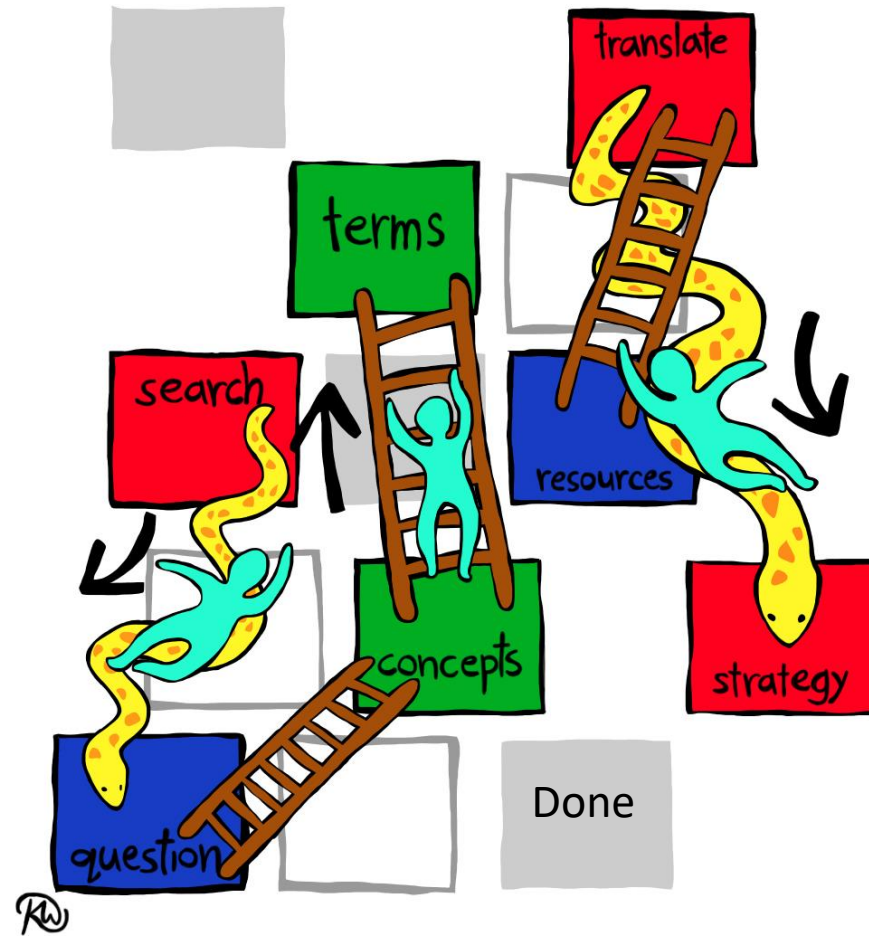
1. BDNF treatment(s)
2. APP processing enzymes
3. Biochemical molecular changes
4. Behavioral/functional improvements

What are the main concepts?

...if BDNF treatment alone results in similar changes to APP processing enzymes as endurance training and if any observed biochemical molecular changes are accompanied by behavioral/functional improvements.

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“...if BDNF treatment alone results in similar changes to APP processing enzymes as endurance training and if any observed biochemical molecular changes are accompanied by behavioral/functional improvements”

1. BDNF treatment(s)
2. APP processing enzymes
3. Biochemical molecular changes
4. Behavioral/functional improvements

1. “brain-derived neurotrophic factor” or BDNF* or neurotrophin*
2. “amyloid precursor protein*” or APP* or “processing enzyme*”
3. Neurodegenerative or Alzheimer* or “neuronal” or “cognitive impairment*” or neuroinflammation or “synaptic dysfunction*” or neurotoxic*
4. brain or nerve* or cognitive*
4. behav* or function*

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2000+
English language
Neurology and interdisciplinary databases
WofS Complete, PsycINFO, GS

“...if BDNF treatment alone results in similar changes to APP processing enzymes as endurance training and if any observed biochemical molecular changes are accompanied by behavioral/functional improvements”

1. BDNF treatment(s)
2. APP processing enzymes
3. Biochemical molecular changes
4. Behavioral/functional improvements

“brain-derived neurotrophic factor” or BDNF* or neurotrophin*

“amyloid precursor protein*” or APP* or “processing enzyme”

Neurodegenerative or Alzheimer* or “neuronal” or “cognitive impairment*” or neuroinflammation or “synaptic dysfunction*” or neurotoxic*

brain or nerve* or cognitive*

behav* or function*

5. Scholarly articles
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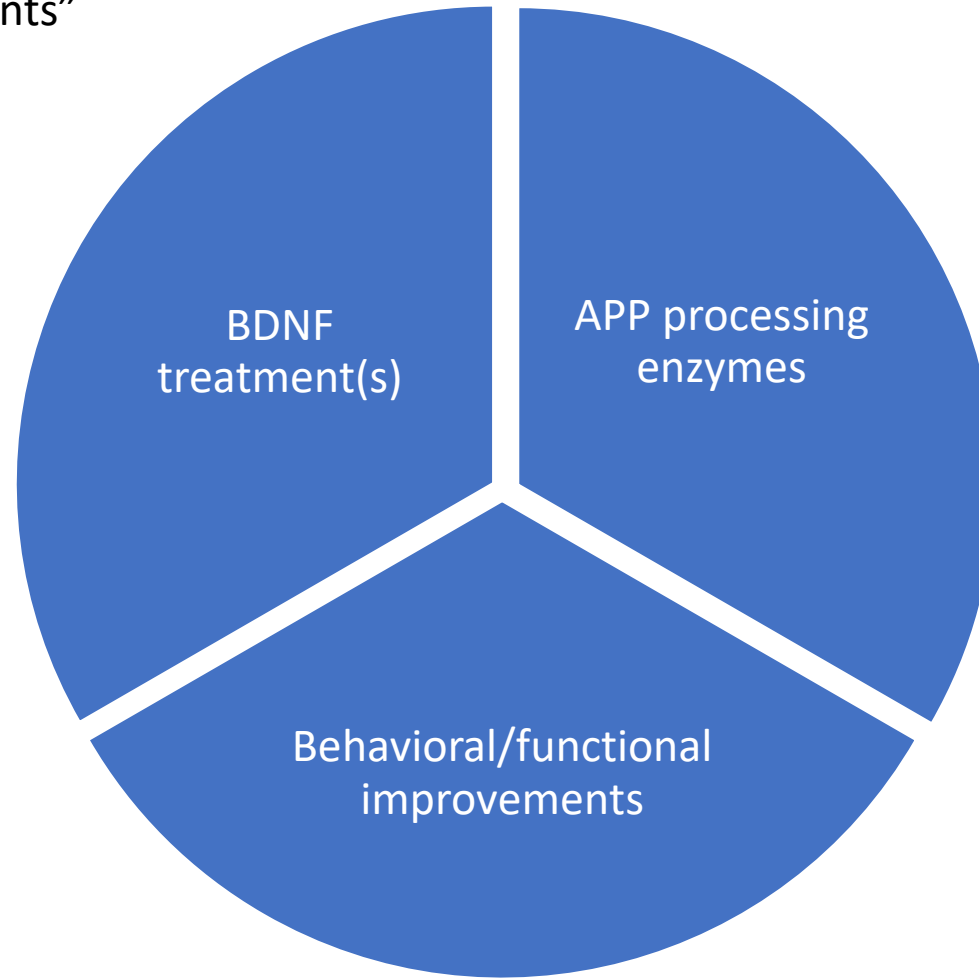
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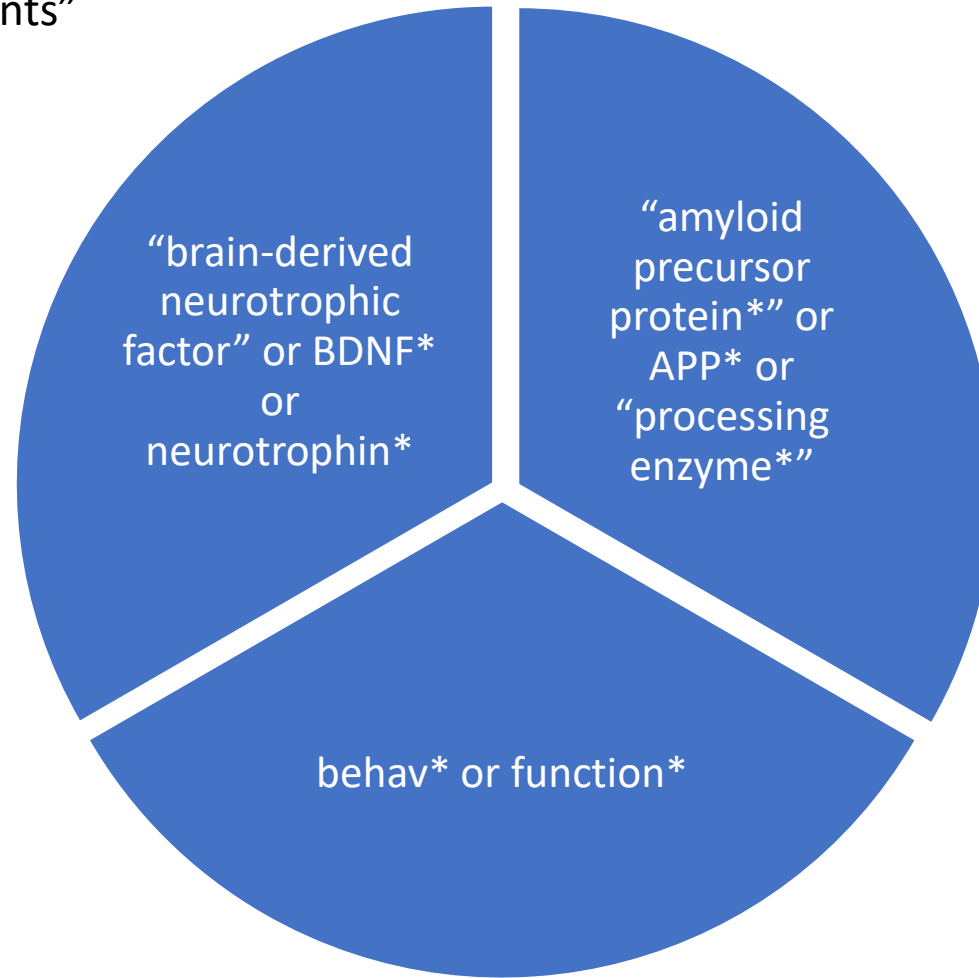
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“...if BDNF treatment alone results in similar changes to APP processing enzymes as endurance training and if any observed biochemical molecular changes are accompanied by behavioral/functional improvements”



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What is another word for behavioral?

Need *synonyms for behavioral*? Here's a list of *similar words* from our *thesaurus* that you can use instead.

Contexts

- Of or relating to observable behavior
- Relating to moral principles or the branch of knowledge dealing with these

Adjective

Of or relating to observable behavior

social comportmental developmental habitual interactive

praxeological etiquettical expressional physiological reactionary

attitudinal manneristic outward practical communicative formal

modal seemly stylistic tonal

*"Common sense applied to the particular **behavioral** quirks of your child is probably your best bet."*

Adjective

Relating to moral principles or the branch of knowledge dealing with these

ethical behavioural^{UK} moral social having to do with right and wrong

moralistic to do with right and wrong



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MeSH

MeSH

alzheimer's disease

Limits

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Alzheimer Disease

A degenerative disease of the BRAIN characterized by the insidious onset of DEMENTIA. Impairment of MEMORY, judgment, attention span, and problem solving skills are followed by severe APRAXIAS and a global loss of cognitive abilities. The condition primarily occurs after age 60, and is marked pathologically by severe cortical atrophy and the triad of SENILE PLAQUES; NEUROFIBRILLARY TANGLES; and NEUROPIL THREADS. (From Adams et al., Principles of Neurology, 6th ed, pp1049-57)

Year introduced: 1998(1963)

PubMed search builder options

Subheadings:

☐ blood	☐ enzymology	☐ pathology
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See Also:

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Alzheimer Disease

Tree Number(s): C10.228.140.380.100, C10.574.945.249, F03.615.400.100

MeSH Unique ID: D000544

Entry Terms:

- Alzheimer Dementia
- Alzheimer Dementias
- Dementia, Alzheimer
- Alzheimer's Disease
- Dementia, Senile
- Senile Dementia
- Dementia, Alzheimer Type
- Alzheimer Type Dementia
- Alzheimer-Type Dementia (ATD)
- Alzheimer Type Dementia (ATD)
- Dementia, Alzheimer-Type (ATD)
- Alzheimer Type Senile Dementia
- Primary Senile Degenerative Dementia
- Dementia, Primary Senile Degenerative
- Alzheimer Sclerosis
- Sclerosis, Alzheimer
- Alzheimer Syndrome
- Alzheimer's Diseases
- Alzheimer Diseases
- Alzheimers Diseases
- Senile Dementia, Alzheimer Type
- Acute Confusional Senile Dementia
- Senile Dementia, Acute Confusional
- Dementia, Presenile
- Presenile Dementia
- Alzheimer Disease, Late Onset
- Late Onset Alzheimer Disease
- Alzheimer's Disease, Focal Onset
- Focal Onset Alzheimer's Disease
- Familial Alzheimer Disease (FAD)
- Alzheimer Disease, Familial (FAD)
- Familial Alzheimer Diseases (FAD)
- Alzheimer Disease, Early Onset
- Early Onset Alzheimer Disease
- Presenile Alzheimer Dementia

Attention Deficit Disorder with Hyperactivity

A behavior disorder originating in childhood in which the essential features are signs of developmentally inappropriate inattention, impulsivity, and hyperactivity. Although most individuals have symptoms of both inattention and hyperactivity-impulsivity, one or the other pattern may be predominant. The disorder is more frequent in males than females. Onset is in childhood. Symptoms often attenuate during late adolescence although a minority experience the full complement of symptoms into mid-adulthood. (From DSM-V)

Year introduced: 1984

Entry Terms:

- Attention Deficit Disorders with Hyperactivity
- ADHD
- Attention Deficit Hyperactivity Disorder
- Hyperkinetic Syndrome
- Syndromes, Hyperkinetic
- Attention Deficit-Hyperactivity Disorder
- Attention Deficit-Hyperactivity Disorders
- Deficit-Hyperactivity Disorder, Attention
- Deficit-Hyperactivity Disorders, Attention
- Disorder, Attention Deficit-Hyperactivity
- Disorders, Attention Deficit-Hyperactivity
- ADDH
- Attention Deficit Hyperactivity Disorders
- Attention Deficit Disorder
- Attention Deficit Disorders
- Deficit Disorder, Attention
- Deficit Disorders, Attention
- Disorder, Attention Deficit
- Disorders, Attention Deficit
- Brain Dysfunction, Minimal
- Dysfunction, Minimal Brain
- Minimal Brain Dysfunction

Previous Indexing:

- [Brain Damage, Chronic \(1966-1968\)](#)

[All MeSH Categories](#)

[Psychiatry and Psychology Category](#)

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Attention Deficit Disorder with Hyperactivity

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⊖ And ▾ Topic ▾ Example: oil spill* mediterranean

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+ BDNF

+ BRAIN DERIVED NEUROTROPHIC FACTOR

+ NEUROTROPHINS

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1 Agent useful for treating choriocarcinoma, comprises inhibitor of brain-derived neurotrophic factor (BDNF), and/or brain-derived neurotrophic factor receptor (TrKB)

JP2012041311-A

Inventor(s) : KAWAMURA K

Assignee(s) : UNIV AKITA

Derwent Primary Accession Number :

2012-C78194

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☐ 1

delta-Opioid receptor as a potential therapeutic target for ischemic stroke

Subedi, K and Wang, HM

Jan 2020 | NEURAL REGENERATION RESEARCH 15 (1) , pp.20-24

Ischemic stroke is a global epidemic condition due to an inadequate supply of blood and oxygen to a specific area of brain either by arterial blockage or by narrowing of blood vessels. Despite having advancement in the use of thrombolytic and clot removal medicine, significant numbers of stroke patients are still left out without option for treatment. In this review, we summarize recent researc

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Example: oil spill* mediterranean

"amyloid precursor protein*" or APP* or "processing enzyme"

✕

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And ▾

Title ▾

Example: water consum*

behav* or function*

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1 High levels of Alzheimer beta-amyloid precursor protein (APP) in children with severely autistic behavior and aggression

Sokol, DK; Chen, DM; (...); Lahiri, DK

Jun 2006 | JOURNAL OF CHILD NEUROLOGY 21 (6) , pp.444-449

Autism is characterized by restricted, repetitive behaviors and impairment in socialization and communication. Although no neuropathologic substrate underlying autism has been found, the findings of brain overgrowth via neuroimaging studies and increased levels of brain-derived neurotrophic factor (BDNF) in neuropathologic and blood studies favor an anabolic state. We examined acetylcholinesterase activity in the brain of children with autism and found significantly higher levels in the brain of children with autism compared to controls. ... Show more

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Quick add keywords: ? <

+ BDNF

+ OXIDO NITROSATIVE STRESS

+ DEPRESSION LIKE BEHAVIORS

+ TRK RECEPTOR

+ BRAIN DERIVED NEUOTRO

>

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[Sokol, DK](#); [Chen, DM](#); (...); [Lahiri, DK](#)

Jun 2006 | [JOURNAL OF CHILD NEUROLOGY](#) 21 (6) , pp.444-449

Autism is characterized by restricted, repetitive behaviors and impairment in socialization and communication. Although no neuropathologic substrate underlying autism has been found, the findings of brain overgrowth via neuroimaging studies and increased levels of brain-derived neurotrophic factor (BDNF) in neuropathologic and blood studies favor an anabolic state. We examined acetylcholinesterase activity in the brains of children with autism spectrum disorders (ASD) and found significantly higher levels of activity in the brains of children with ASD compared to controls. ... [Show more](#)

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1 Neural stem cells improve cognition via BDNF in a transgenic model of Alzheimer disease

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Blurton-Jones, M; Kitazawa, M; (...); LaFerla, FM

Aug 11 2009 | PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA 106 (32) , pp.13594-13599

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References

Neural stem cell (NSC) transplantation represents an unexplored approach for treating neurodegenerative disorders associated with cognitive decline such as Alzheimer disease (AD). Here, we used aged triple transgenic mice (3xTg-AD) that express pathogenic forms of amyloid precursor protein, presenilin, and tau to investigate the effect of neural stem cell transplantation on AD-related neuropathology. ... Show more

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Barha, CK; Davis, JC; (...); Liu-Ambrose, T

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References

Exercise is a non-pharmacological strategy to mitigate the deleterious effects of aging on brain health. However, a large amount of variation exists in its efficacy. Sex of participants and exercise type are two possible factors contributing to this variation. To better understand this, we conducted a concurrent systematic review and meta-analysis of cognitively healthy older adults. Executive ... Show more

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Title	Creator
Aerobic Exercise Improves Cognition for Older Adults with Glucose Intolerance, A Risk Factor for Alzheimer's Disease	Baker et al.
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Exercise training and BDNF injections alter amyloid precursor protein (APP) processing enzymes and improve cognition	Baranowski et al.
Exercise training and BDNF injections alter amyloid precursor protein (APP) processing enzymes and improve cognition	Baranowski et al.
FGF19 improves sevoflurane-induced cognitive dysfunction in rats through the PGC-1 alpha/BDNF/FNDC5 pathway	Lu et al.
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Synergistic Increase of Serum BDNF in Alzheimer Patients Treated with Cerebrolysin and Donepezil: Association with Co

The Oxford companion to The body

Whole-brain low-intensity pulsed ultrasound therapy markedly improves cognitive dysfunctions in mouse models of d

Creator

Baker et al.

Ji et al.

Wu et al.

Willeumier and Toland

Wood et al.

Augusto-Oliveira et

Create Citation/Bibliography

Citation Style:

American Chemical Society

American Medical Association 11th edition

American Political Science Association

American Psychological Association (APA) 7th edition

American Psychological Association (APA) 7th edition (with abstract)

American Sociological Association (ASA) 6th edition

Chicago Manual of Style 17th edition (author-date)

Chicago Manual of Style 17th edition (full note)

Language:

English (US)

Output Mode:

Citations

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Calibri (Body) 11 A⁺ A⁻ Aa A₁

B I U x₂ x² A₁ A₂ A₃

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Alvarez, X., Alvarez, I., Iglesias, O., Crespo, I., Figueroa, J., Aleixandre, M., Linares, C., Granizo, E., Garcia-Fantini, M., Marey, J., Masliah, E., Winter, S., Muresanu, D., & Moessler, H. (2016). Synergistic Increase of Serum BDNF in Alzheimer Patients Treated with Cerebrolysin and Donepezil: Association with Cognitive Improvement in ApoE4 Cases. *INTERNATIONAL JOURNAL OF NEUROPSYCHOPHARMACOLOGY*, 19(6). <https://doi.org/10.1093/ijnp/pyw024>

Augusto-Oliveira, M., Arrifano, G., Leal-Nazare, C., Santos-Sacramento, L., Lopes-Araujo, A., Royes, L., & Crespo-Lopez, M. (2023). Exercise Reshapes the Brain: Molecular, Cellular, and Structural Changes Associated with Cognitive Improvements. *MOLECULAR NEUROBIOLOGY*. <https://doi.org/10.1007/s12035-023-03492-8>

Baker, L., Frank, L., Foster-Schubert, K., Green, P., Wilkinson, C., McTiernan, A., Cholerton, B., Plymate, S., Fishel, M., Watson, G., Duncan, G., Mehta, P., & Craft, S. (2010). Aerobic Exercise Improves Cognition for Older Adults with Glucose Intolerance, A Risk Factor for Alzheimer's Disease. *JOURNAL OF ALZHEIMERS DISEASE*, 22(2), 569–579. <https://doi.org/10.3233/JAD-2010-100768>

This is an essay about BDNF as it applies to APP-related behaviors and functions within specific populations (Alvarez, X. et al., 2016; Baranowski et al., 2023; Gordon, 2013; Vedovelli et al. 2017) as part of a research team. This research supports the claim...

References

Alvarez, X., Alvarez, I., Iglesias, O., Crespo, I., Figueroa, J., Aleixandre, M., Linares, C., Granizo, E., Garcia-Fantini, M., Marey, J., Masliah, E., Winter, S., Muresanu, D., & Moessler, H. (2016). Synergistic Increase of Serum BDNF in Alzheimer Patients Treated with Cerebrolysin and Donepezil: Association with Cognitive Improvement in ApoE4 Cases. *INTERNATIONAL JOURNAL OF NEUROPSYCHOPHARMACOLOGY*, 19(6). <https://doi.org/10.1093/ijnp/pyw024>

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Baranowski, B. J., Mohammad, A., Finch, M. S., Brown, A., Dhaliwal, R., Marko, D. M., LeBlanc, P. J., McCormick, C. M., Fajardo, V. A., & MacPherson, R. E. K. (2023). Exercise training and BDNF injections alter amyloid precursor protein (APP) processing enzymes and improve cognition. *Journal of Applied Physiology*, 135(1), 121–135. <https://doi.org/10.1152/jappphysiol.00114.2023>



Controversies on the use of eponyms in neurosciences.

Cognitive Neuroscience

Field of study concerning MENTAL PROCESSES; LEARNING; and the brain regions associated with them.

Year introduced: 2016

PubMed search builder options

[Subheadings:](#)

- | | | |
|---|--|--|
| ☐ classification | ☐ history | ☐ organization and administration |
| ☐ economics | ☐ instrumentation | ☐ standards |
| ☐ education | ☐ legislation and jurisprudence | ☐ statistics and numerical data |
| ☐ ethics | ☐ methods | ☐ trends |

☐ Restrict to MeSH Major Topic.

☐ Do not include MeSH terms found below this term in the MeSH hierarchy.

Tree Number(s): F04.096.628.255.500, H01.158.610.030

MeSH Unique ID: D000066494

Entry Terms:

- Neuroscience, Cognitive
- Social Neuroscience
- Neuroscience, Social
- Neurosciences, Social
- Social Neurosciences

[All MeSH Categories](#)

[Psychiatry and Psychology Category](#)

[Behavioral Disciplines and Activities](#)

[Behavioral Sciences](#)

[Psychology](#)

[Cognitive Science](#)

Cognitive Neuroscience

[All MeSH Categories](#)

[Disciplines and Occupations Category](#)

[Natural Science Disciplines](#)

[Biological Science Disciplines](#)

[Neurosciences](#)

Cognitive Neuroscience

Neurology

A medical specialty concerned with the study of the structures, functions, and diseases of the nervous system.

PubMed search builder options

Subheadings:

- | | | |
|---|--|--|
| ☐ classification | ☐ history | ☐ organization and administration |
| ☐ economics | ☐ instrumentation | ☐ standards |
| ☐ education | ☐ legislation and jurisprudence | ☐ statistics and numerical data |
| ☐ ethics | ☐ methods | ☐ trends |

☐ Restrict to MeSH Major Topic.

☐ Do not include MeSH terms found below this term in the MeSH hierarchy.

Tree Number(s): H02.403.600

MeSH Unique ID: D009462

See Also:

- [Nervous System Diseases](#)

[All MeSH Categories](#)

[Disciplines and Occupations Category](#)

[Health Occupations](#)

[Medicine](#)

Neurology

[Neuropathology](#)

[Neurotology](#)

Eponyms

Names based on or derived from the name of a person or a place.

PubMed search builder options

- ☐ Restrict to MeSH Major Topic.
- ☐ Do not include MeSH terms found below this term in the MeSH hierarchy.

Tree Number(s): L01.559.598.400.556.536

MeSH Unique ID: D004850

Entry Terms:

- Eponym

[All MeSH Categories](#)

[Information Science Category](#)

[Information Science](#)

[Language](#)

[Linguistics](#)

[Terminology as Topic](#)

[Names](#)

Eponyms

Controversies on the use of eponyms in neurosciences.

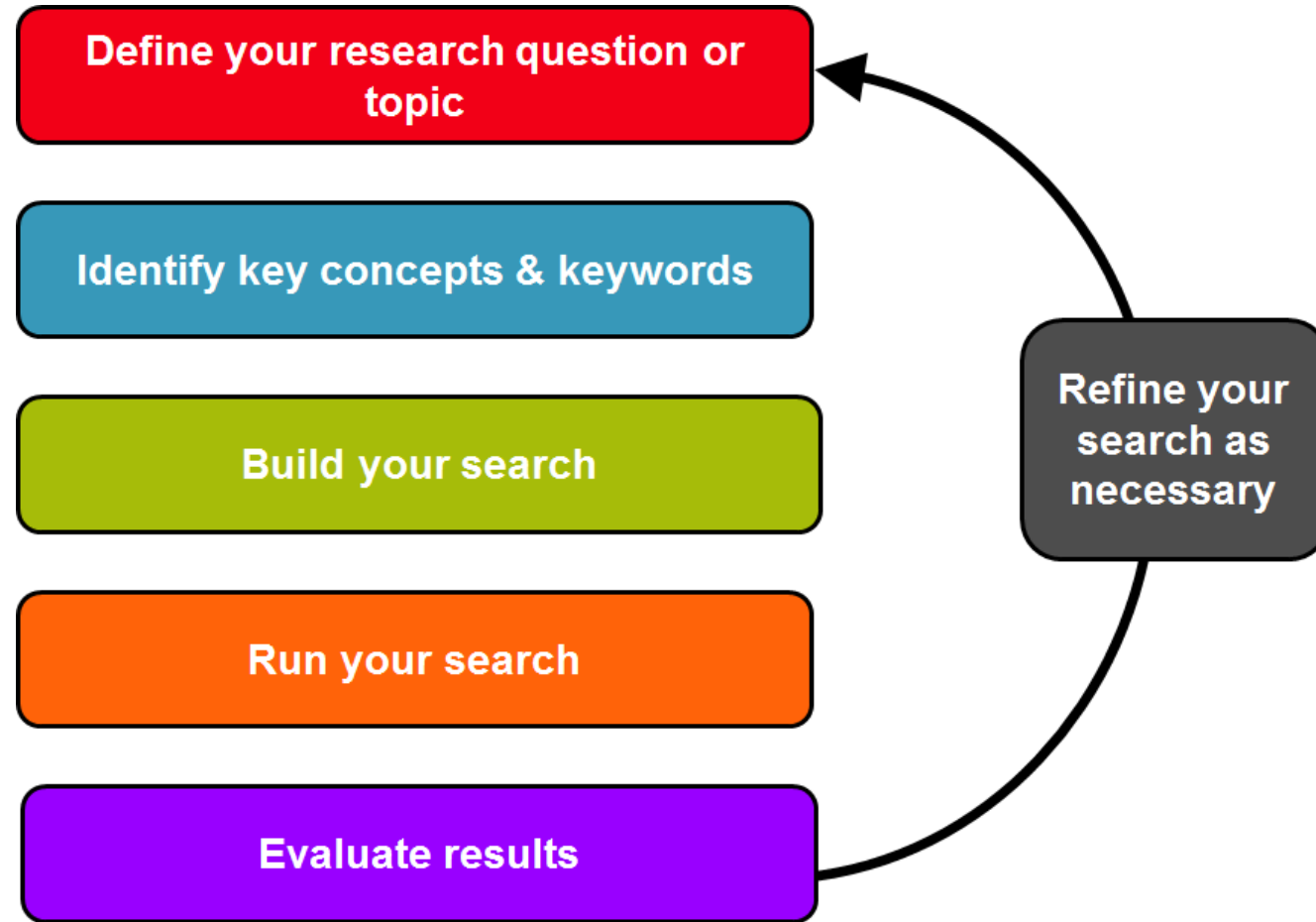
Search Google Scholar and critically evaluate and share your findings

Controversies on the use of eponyms in neurosciences.

Search Web of Science complete and critically evaluate and share your findings

Library Seminar Agenda

- Top 10 resources you should know about!
- What is neuroscience?
- Scholarly resources – books
- Scholarly resources – databases
- Scholarly resources – articles
- [How to search for information](#)
- Where, how and when to get help!
- Questions / Answers



Neuroscience tips and tricks

Look for seed articles

Identify main concepts

Look for alternate terms

Consult thesauri e.g. MeSH, Wordhippo

Define terms

Select database(s)

Create database-specific search strategies

Think like the database when searching

Search, refine, filter and browse...

Cite, Zotero, send to Zotero

Refine

Never too early to start

Be open to change

Ask for assistance

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Where can I get help?

Brock Library Neuroscience Research Guide

<https://researchguides.library.brocku.ca/NEUR>

Email the Library

libhelp@brocku.ca

Ask Us Chat service

<https://brocku.ca/library/chat/>

Ask Us Chat 



Book a Consultation

<https://calendar.library.brocku.ca/appointments/researchconsultation>

Make an Appointment Research Consultation

Your Librarian can help you:

- use the best search tools for your assignments
- find information sources on your specific topic
- develop effective research strategies
- become a confident and independent researcher

1. Select One

▼ Librarian

Research Consultation (30 minutes) ▼

☐ Justine Cotton



☒ Ian Gordon (he/him)



☐ Elizabeth Yates



☐ Kymberly Ash



☐ Denise Smith



2. Select Date:

🕒

Sep ▼

2023

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Su	Mo	Tu	We	Th	Fr	Sa
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3	4	5	6	7	8	9
10	11	12	13	14	15	16
17	18	19	20	21	22	23
24	25	26	27	28	29	30

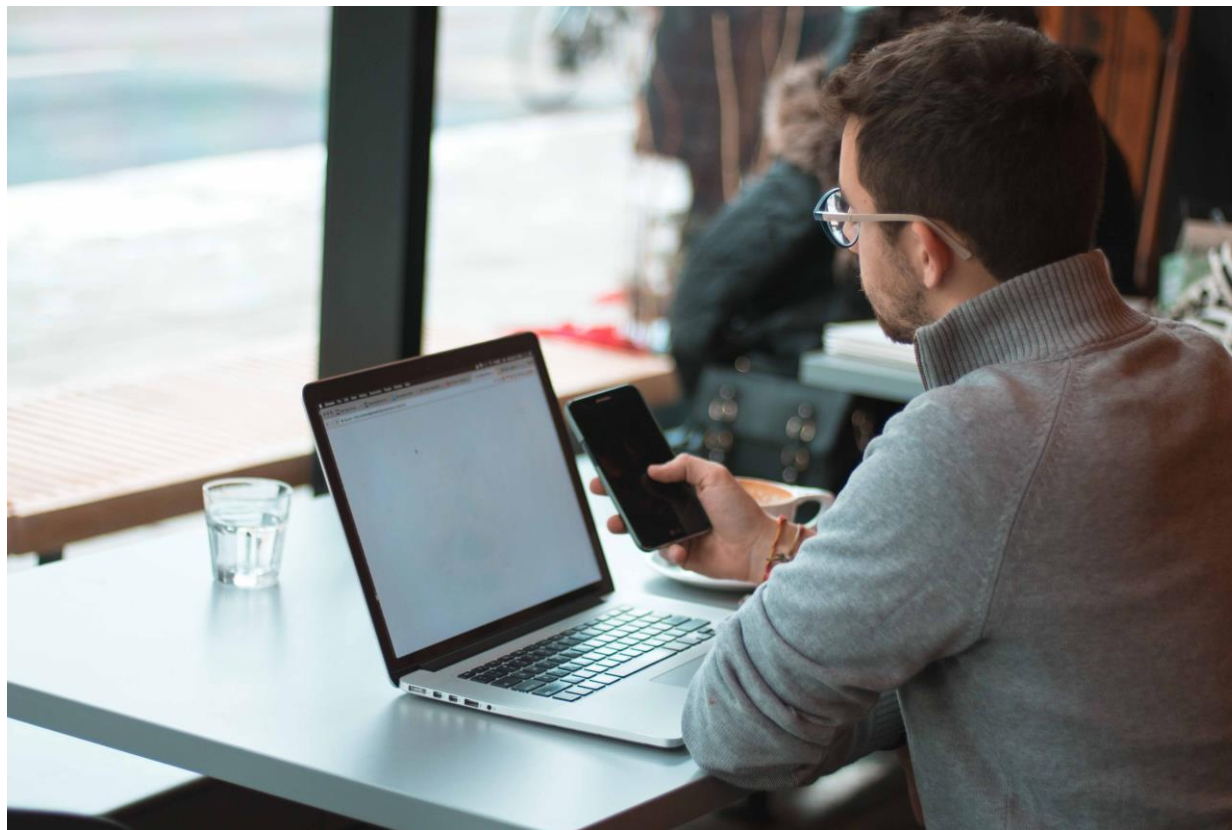
3. Select Time:

Friday, September 29, 2023

Time Zone: Eastern Time - US & Canada ([change](#))

9:03am	9:33am	10:03am
10:33am	11:03am	11:33am
12:03pm	12:33pm	1:03pm
1:33pm	2:03pm	2:33pm
3:03pm	3:33pm	

Continue



Man sitting near table with laptop photo
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Top 10 resources you should know about!

1. Zotero
2. Zotero
3. Omni
4. Borrowing from Other Libraries
5. Brock Library NEUR Library Research Guide
6. Google Scholar, Google Books and Advanced Google searching
7. Open Education Resources (OERs)
8. PubMed / MEDLINE
9. When, where and how to get help!
10. Book a Consultation

One thing you've learned today?



Ian Gordon

Teaching & Learning Librarian
igordon@brocku.ca



All the best with your assignments,
writing, research and learning.

Library Seminar Agenda

- Top 10 resources you should know about!
- What is neuroscience?
- Scholarly resources – books
- Scholarly resources – databases
- Scholarly resources – articles
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